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# Phytopathology

Official Organ of the

## American Phytopathological Society

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PHYTOPATHOLOGY

VOLUME X NUMBER 2

FEBRUARY, 1920

# A BACTERIAL LEAFSPOT OF VELVET BEAN

FREDERICK A. WOLF

WITH TWO FIGURES IN THE TEXT

During the summer of 1916, a leafspot of Florida velvet bean, *Stizolobium deeringianum* Bort, was noted in the vicinity of West Raleigh, N. C. It was apparently unlike any known disease affecting this crop, as judged from available published accounts. Furthermore, preliminary examination of these specimens showed that the affected areas were teeming with bacteria. Since the foliage of soybeans, *Soja max*, growing in the fields together with velvet beans was affected with a bacterial blight, it was thought at first that the causal organism on both host species was probably identical. Furthermore the colonies in the isolations on nutrient agar looked so similar that it did not become apparent that one was dealing with two distinct organisms until morphological studies were made and reciprocal inoculations were attempted. Parallel studies of the morphology, of the cultural and biochemical characters, and of the pathogenicity of the velvet bean leafspot organism and soybean bacterial blight, *Bacterium sojae*[1] have therefore been made, and the results of the studies on the former are given herewith whereas those on the latter are presented in another paper appearing in the March number of this Journal.

## THE DISEASE

The disease has been observed to be more or less destructive during the past four years, not only in the vicinity in which it was first seen, but specimens have also been collected in several widely separated portions of the state. In no case has it been sufficiently severe to be a limiting factor in the culture of velvet beans. Furthermore, since the variety

[1] Coerper, Florence M. Bacterial Blight of Soybean. In Jour. Agr. Research **18**: no. 4, pp. 179–193, pls. A and 12–18. 1919.

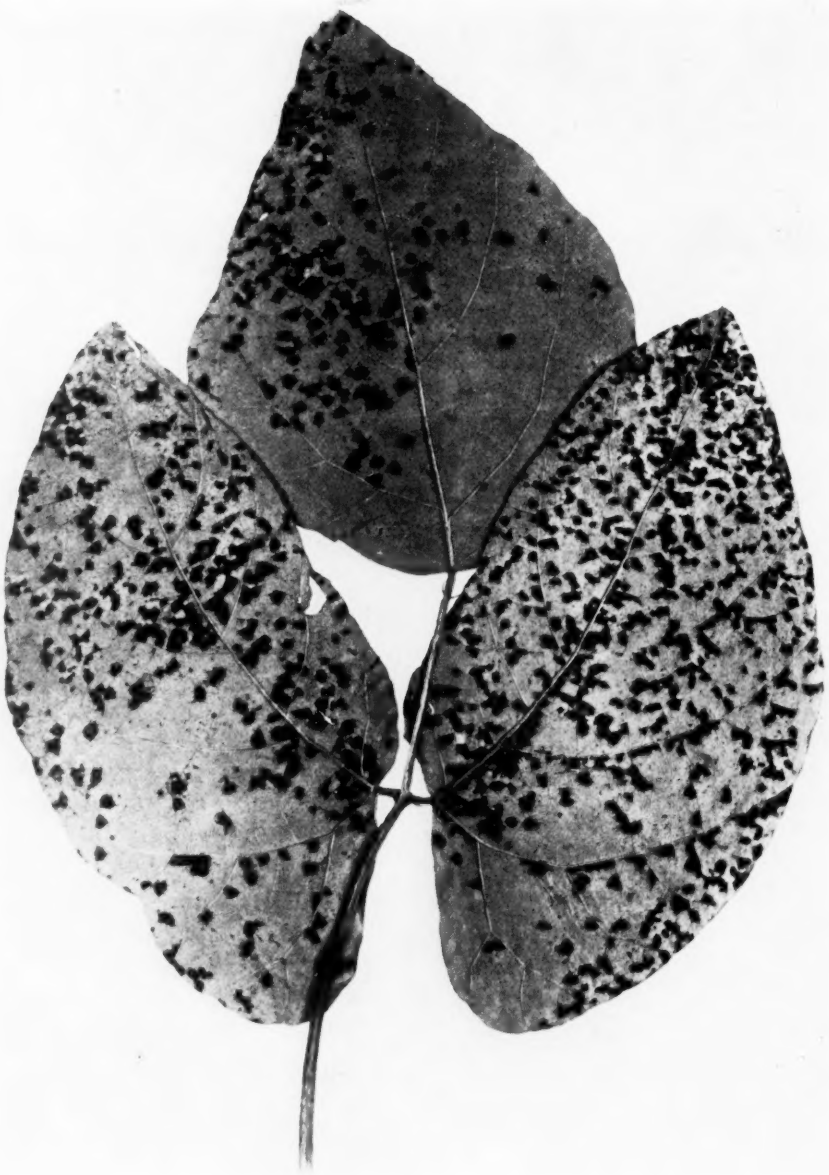

FIG. 1. LOWER LEAF SURFACE OF FLORIDA VELVET BEAN LEAF SHOWING THE APPEARANCE OF BACTERIAL LEAFSPOT

known as "Hundred Day Speckle" alone will properly mature in the latitude of North Carolina, no opportunity has been given to make field observations upon the susceptibility of Lyon bean, *Stizolobium niveum* (Roxb.) Kuntze, and Yokohama bean, *Stizolobium hassjoo* Piper and Tracy, species grown farther southward.

The disease may occur in fields which have never previously been planted to velvet beans, indicating transmission of the disease through seed. Apparently, however, the foliage only is subject to attack. Here the disease is first evident by the presence of small, point-like, translucent areas. The centers of these areas early become dark brown, and the translucent character largely disappears except at the extreme periphery. The water-soaked border, so characteristic of bacterial leaf spots, is entirely absent in mature lesions. An area near the center of mature spots is characteristically lighter in color than the margin. The tissues immediately surrounding the invaded areas are more or less chlorotic, and this chlorosis may involve all of the leaf surface not occupied by lesions. Mature spots are angular, being limited by the veins except when they are so abundant as to merge into large, irregular areas. They are commonly 2 to 3 mm. across, although they vary in size from mere points to spots 8 mm. in width. Severely diseased leaves may have as many as several hundred spots on each leaflet, as shown in figure 1. Little difference in appearance is to be noted between the symptoms presented by the upper and lower leaf surface, and no bacterial exudate is manifest.

## THE ORGANISM

The organism being non-motile is referred to the genus Aplanobacter, and since it is apparently unlike any previously described organism the specific name stizolobii is suggested. It is therefore described as *Aplanobacter stizolobii* n. sp.

## MORPHOLOGY

*Vegetative cells.* The organism is a short rod with rounded ends. It occurs singly or in pairs when taken from the host and may form short chains in nutrient broth. When taken from nutrient agar cultures and stained with carbol fuchsin it is slightly variable in length, being 1 to 1.6 by 0.6 to 0.7 $\mu$ (fig. 2, *b*). Mature elements average 1.3 $\mu$ in length and individuals exceeding this measurement appear to be undergoing fission.

*Endospores.* No endospores have been observed and little probability of their occurrence exists, in the light of the fact that the thermal death point is not far from 50 degrees C.

*Flagella.* Attempts to demonstrate flagella have been without success. Flagella of *Bacterium sojæ* are sharply differentiated by Morrey's modification of Loeffler's method, whereas no flagella are demonstrable on the velvet bean organism upon the opposite end of the same slide, where it is subjected to the same conditions of staining. When it is taken from young lesions and from 24-hour cultures on nutrient agar or in broth and examined for motility no evidence of independent movement can be discerned.

*Capsules.* The organisms used for this purpose were grown on potato plugs and were stained by the method of Räbiger and of Welch. A conspicuous envelope may be demonstrated by either of these methods (fig. 2, *a*).

*Zoogloeae.* Neither zoogloeae nor pseudozoogloeae are formed.

*Involution forms.* None observed.

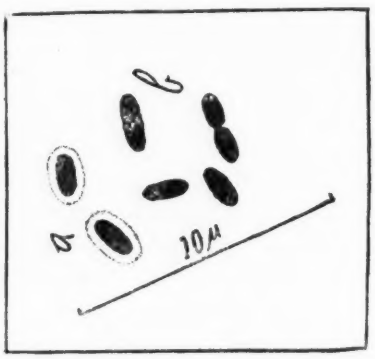


FIG. 2. *a*, APLANOBACTER STIZOLOBII STAINED BY WELCH'S METHOD; *b*, STAINED WITH CARBOL FUCHSIN

*Staining reactions.* Efforts at staining with the ordinary stains in both watery and alcoholic solutions have been uniformly successful. Gram's stain and Zeihl-Neelson's, however, proved negative.

## CULTURAL CHARACTERS

No particular difficulty has been experienced in isolating the parasite. Lesions from freshly collected material were cut out and thoroughly macerated in a drop of sterile water on a microscopic slide. A platinum loopful of this material was transferred to one edge of a hardened plate of nutrient agar, neutral to phenolphthalein. The material was then spread over the surface of the agar plate with a zigzag stroke in the ultimate direction of the side of the plate opposite to the one where the inoculum was deposited. The organisms are so distributed by this manner of

spreading that well isolated colonies develop near the end of the stroke. The plate, of course, always contains species other than the parasite when this method of isolation is employed, since no attempt is made to disinfect the leaf surface before excision of lesions. The parasite uniformly predominates, however, and the danger of its destruction within the tissues, as so commonly occurs from the use of alcohol or bichlorid of mercury, is eliminated.

The peptone beef-extract agar and bouillon used in the cultural studies were enriched by the addition of either dextrose, lactose, glycerin, or saccharose to make 1 per cent solutions. The carbohydrate solutions were prepared separately and added to the media when the temperature had been lowered to about 60°C., and the media were not heated subsequently. All of the various media used in one series of cultural studies were inoculated on the same day from the same stock culture.

*Agar colonies.* When isolations are made from diseased tissue the colonies appear on the second day and are convex, semitransparent, circular, shining, and minute. By the fourth or fifth day at 20 to 25°C. they have attained their maximum size and are 1 to 1.5 mm. in diameter. They have a shiny luster, are smooth, white, raised, and opaque, and possess an entire or slightly undulate margin.

On agar slants there is a moderate filiform growth which spreads at the base of the slant where moisture of condensation is present. The streak is whitish and does not penetrate the agar nor give rise to any perceptible odor.

In agar stabs the growth is confined to the surface. It covers the greater portion of the tube in the course of a month, the edges are contoured, and there is no liquefaction of the agar.

*Gelatin colonies.* Stab cultures only, maintained at about 18°C., were used. There was no evidence of growth along the line of the stab and no liquefaction at the end of thirty days. The organism was able to make sufficient growth in this time, however, to cover the surface of the tube.

*Potato cylinders.* When the surface of the cylinders is very moist, growth is moderate, white, glistening, and spreading, whereas rather dry plugs have a raised, lusterless growth. The upper end of the streak will have become of a cheesy consistency within a month. No odor is developed within this period, the substratum does not become discolored, and there is no evidence of diastatic action when tested by means of iodin in potassium iodid solution. The organism is without effect on corn starch agar.

*Bouillon.* Peptone beef-extract bouillon becomes only slightly clouded throughout; no pellicle, ring, nor odor is developed.

*Litmus milk.* Freshly separated milk, without neutralizing, was made up to a violaceous color and subjected to four sterilizations in an Arnold sterilizer, at intervals of 12 hours. After ten days the blue color is increased to slightly darker than caeruleus. A considerable white precipitate appears within a month and the color is unchanged.

### PHYSICAL AND BIOCHEMICAL CHARACTERS

In the studies on reaction toward the several sugars, use was made of stab cultures on litmus agar and of peptone broth in fermentation tubes to both of which the appropriate carbon compounds had been added in the manner previously described.

*Litmus agar.* Neither acid nor gas were formed from glycerin, lactose, saccharose, or dextrose, although the cultures were incubated at about 20°C. for thirty days.

*Fermentation tubes.* Triplicate sets of fermentation tubes were prepared, inoculated, and kept under observation for three weeks. The open arm in all fermentation tubes became slightly clouded but in no case did growth extend into the closed arm, indicating strict aerobism. Neither was gas formed in the closed arm of any of the tubes.

*Nitrates.* Five-day-old cultures in 1 per cent potassium nitrate bouillon solution were tested for nitrites by the starch-iodide method. No blue color resulted, which indicates that there was no reduction of nitrates to nitrites. A solution of sodium nitrite used to test the reagents gave the characteristic reaction, however.

*Indol.* A few drops of concentrated sulphuric acid and 0.01 per cent solution of potassium nitrite were added to ten-day-old cultures in Dunham's solution. Even after the tubes were gently heated no pink color developed, indicating the absence of indol.

*Temperature relations.* The optimum temperature for the cultivation of this organism has not been carefully determined but appears to lie between 25 and 28°C. The thermal death point determined by exposing newly inoculated bouillon cultures for ten minutes to definite temperatures appears to be not far from 50°C.

*Group number.* Following the numerical system of the Society of American Bacteriologists the group number of this organism is 212.3332033.

### COMPARISON OF BACTERIUM SOJÆ AND APLANOBACTER STIZOLOBII

Since, as has been indicated, these two organisms looked so similar when the first isolations were made, parallel studies on one strain each, according to the essential features on the descriptive chart, were made.

The results of this comparison indicate that these strains differ strikingly only as follows:

| ORGANISM | SIZE | FLAGELLA | DIASTATIC ACTION | GROWTH IN BOUILLON |
|---|---|---|---|---|
| *Bacterium sojæ*..... | 1.2–2.6 by 0.8–0.9 $\mu$ | 1 to 4 | Very feeble | Surface pellicle and moderate clouding. |
| *Aplanobacter stizolobii*..... | 1–1.6 by 0.6–0.7 $\mu$ | None | None | No pellicle with less dense clouding |

## SUMMARY OF DESCRIPTIVE CHARACTERS

The results of the studies on the identity of the velvet bean leafspot organism may be briefly summarized as follows:

**Aplanobacter stizolobii** n. sp.

Elements cylindrical with rounded ends, single or in pairs, 1–1.6 by 0.6–0.7 $\mu$, non-motile, capsulate, no endospores, zoogloeae not formed. Neither acid fast nor stained by Gram's method.

Colonies on nutrient agar circular, raised, white, shining, margin entire or slightly undulate.

Gelatin not liquefied; casein partially precipitated and blue color in litmus milk intensified; nitrates not reduced; indol not formed; neither acid nor gas formed in cultures with various sugars; no growth in closed arm of fermentation tubes; no diastatic action.

Pathogenic on foliage of *Stizolobium deeringianum*, forming leafspots which are at first translucent and become at maturity dark brown.

Type specimens and artificially inoculated leaves have been deposited in the herbarium of the Office of Mycological Collections, Bureau of Plant Industry, Washington, D. C.

The brief Latin diagnosis is as follows:

Baculis cylindraceis, apicibus rotundatis, solitariis aut interdum geminis, 1-1.6 by 0.6–0.7 $\mu$, non-motilibus, capsulatis, sporis nondum visis; zoogloeis defectis; acidibus decoloratis, cum methodo Grami non coloratis; coloniis in agar-agar orbicularibus, convexis, albidis, nitentibus, marginis solidis aut lobulatis; gelatinis non liquefacientibus; casein ex parte segregantibus; lactem litmus non decolorantibus; nitrum non reducentibus; indol non producentibus; in mediis saccharatis neque gas neque acidum evolvatis; aerobicis; amylum non fermentatis.

Hab. In foliis vivis Stizolobii deeringiani, laesiones primum translucentibus diende-brunneolis efficiens.

## PATHOGENICITY

Florida velvet beans and Mammoth Yellow soybeans are the only host species which have been used in the inoculation experiments. It has been impossible thus far to bring about infection on the latter host

with pure cultures of *Aplanobacter stizolobii*, but characteristic lesions follow inoculation of velvet beans. The following procedure and results are typical of the tests which have been made. A few leaves on one side of a large vine growing in the greenhouse were atomized with a forty-eight-hour bouillon culture late in the afternoon of September 11. On the fifth day following, numerous water-soaked lesions had formed which on the fourteenth day were characteristic of the disease. The abundance of the diseased areas on these artificially inoculated leaves can be judged from the following counts made on ten leaflets taken at random; 31, 215, 108, 35, 101, 175, 52, 96, 109, 30.

An organism which presents the same morphological features and appearance on agar as the original organism has been reisolated from artificial inoculations.

## PATHOLOGICAL HISTOLOGY

Four-day-old and two-week-old lesions from artificial inoculation, and mature lesions from field collections have been fixed, embedded in paraffin, sectioned, and then stained by Loeffler's methylene blue method. The presence of bacteria within the tissues may be clearly demonstrated in velvet bean lesions by means of this method. As in the case of a considerable number of leaf-invading bacteria, entrance is gained through the stomates, and in young lesions the substomatal cavities and adjacent intercellular spaces alone are occupied. As the lesions mature, however, and the invaded tissues become desiccated, the cell walls are disrupted, and the organism comes not only to occupy the intercellular spaces but to fill the interior of the cells. Apparently it is impossible for the organism to attack tissues other than parenchyma.

## SUMMARY

Bacterial leafspot of Florida velvet beans appears to be a hitherto undescribed disease, which has been under observation in North Carolina since 1916.

The disease is apparently restricted to the leaves, on which it is first apparent as translucent areas which become at length dark brown in color.

The parasite is unlike the soybean bacterial blight organism, with which it has been compared in essential characters by means of parallel cultural studies. It is herein described as *Aplanobacter stizolobii*, n. sp., is white in culture, and according to the numerical system has the number 212.3332033.

An incubation period of about four days follows inoculation of uninjured leaves. Infection occurs through the stomata; invasion is first intercellular but later becomes intracellular and only parenchyma tissues are involved.

# HEART ROT, RIB ROT, AND LEAF SPOT OF CHINESE CABBAGES

NELLIE A. BROWN AND R. B. HARVEY

WITH FOUR FIGURES IN THE TEXT

## GENERAL FEATURES

A rot which destroys a high percentage of the plants has been found prevalent on Chinese cabbage varieties in Maryland during the damp weather of last summer and continuing during the unusually warm autumn of this year (1919). The high temperature and wet season are no doubt the cause of the prevalence of the diseased condition. However, some growers have complained of this rot in more normal years and found the production of Chinese cabbages difficult on account of their susceptibility to the rot.

First indications of the disease are given by a sudden wilting of the plants although they are supplied with abundant moisture. In handling such plants it will be found that the head can be easily picked out from the crown of leaves, displaying a very rotten and foul smelling hole in the pith (fig. 1). The tissue within the ring of vascular bundles is usually soft and may be almost entirely removed. The vascular ring is browned. Flies and other insects are attracted by the foul odor, and probably play a part in dissemination of the disease.

A characteristic appearance which may occur accompanied or not by wilting is a browning of the midribs; this occurs noticeably in the looser leaved varieties, Pak choi, *Brassica chinensis* Jusl. Besides this very marked browning of the midrib, specking or spotting of the leaf blade may occur. These spots can be distinguished readily from spots on the leaf caused by *Alternaria brassicae* (Berk.) Sacc., which are round or oval, black spots marked with concentric zones. The Alternaria spot (fig. 2) generally does not cause as much injury as the heart and rib rots, since it attacks only the outer leaves and is more amenable to control by spraying.

All varieties of Chinese cabbage which were tested, including Paoting, Peking, Kinshiu, Chosen, Shantung, and Santo Sai, are subject to this bacterial rot, but it seems more prevalent on the firm-headed varieties, Paoting and Peking of the Pai Ts'ai type. Loose-headed varieties of the

Pak choi type are less susceptible, probably because less moisture collects in the head or owing to the toughness of the epidermis.

When the crown of a diseased plant is cut across it is found to be hollow, the pith gone or nearly so, with the brown-colored vascular tissue left in a sort of honeycomb arrangement. The main and lateral roots are affected similarly, many of them being a brown color throughout. There is usually no soft rot condition of the main root or lateral roots, their affection being a dry rot. The soft rot occurs in the crown and bud, which become a slimy mass. These soft parts are well invaded by nematodes, fungi and bacteria.

The browned midrib is a dry rot (fig. 3) though it is not unusual for the soft stage to occur here also.

## THE CAUSE OF THE DISEASE

After repeated poured-plate isolations from different parts of the plant and from several plants, and inoculations into Chinese cabbage plants, we conclude that the beginning stage of the disease is produced by *Bacterium campestre* (Pam.) Smith, an organism producing the black rot of common cabbage and other cruciferous plants (3), (5), (6). This disease affecting the Chinese cabbages, which are succulent plants and rapid growers, produces numerous lesions which give opportunity for the entrance of a soft rot organism. The latter organism we found present in all the soft parts of the diseased plants. It is not present in the brown midribs unless the soft condition obtains, nor in the early stages of the dry rot condition of the lateral roots.

This soft rot organism seems to be the more destructive of the two (1). While *Bact. campestre* is slowly plugging up the vessels and dissolving the middle lamellae, causing wilting, formation of cavities, and brown staining, should the soft rot germ gain an entrance and be given the proper temperature and moisture conditions its destructive action is so rapid that it can do its work in advance of the wilting and browning. It produces a heart rot which spoils the plant entirely for either sale or home use.

## THE PARASITE

The initial cause of the disease is a short, yellow rod with one polar flagellum, motile but not always so. The rods are usually one and one-half to three times longer than wide, while sometimes they are so short that they resemble coccus forms. The organism most frequently occurs singly, but it occurs also in pairs and occasionally in chains of from 6 to 12 or more of the very small rods. No spores were seen.

These morphological features are exactly like those of *Bact. campestre* isolated from the common cabbage.

FIG. 1. Chinese cabbage disease showing browning of midrib produced by *Bacterium campestre* and soft rot at heart produced by a soft rot organism.

FIG. 2. Chinese cabbage leaf showing browning of midrib and small dark spots produced by *Bacterium campestre;* also zoned fungous spots of *Alternaria brassicae.*

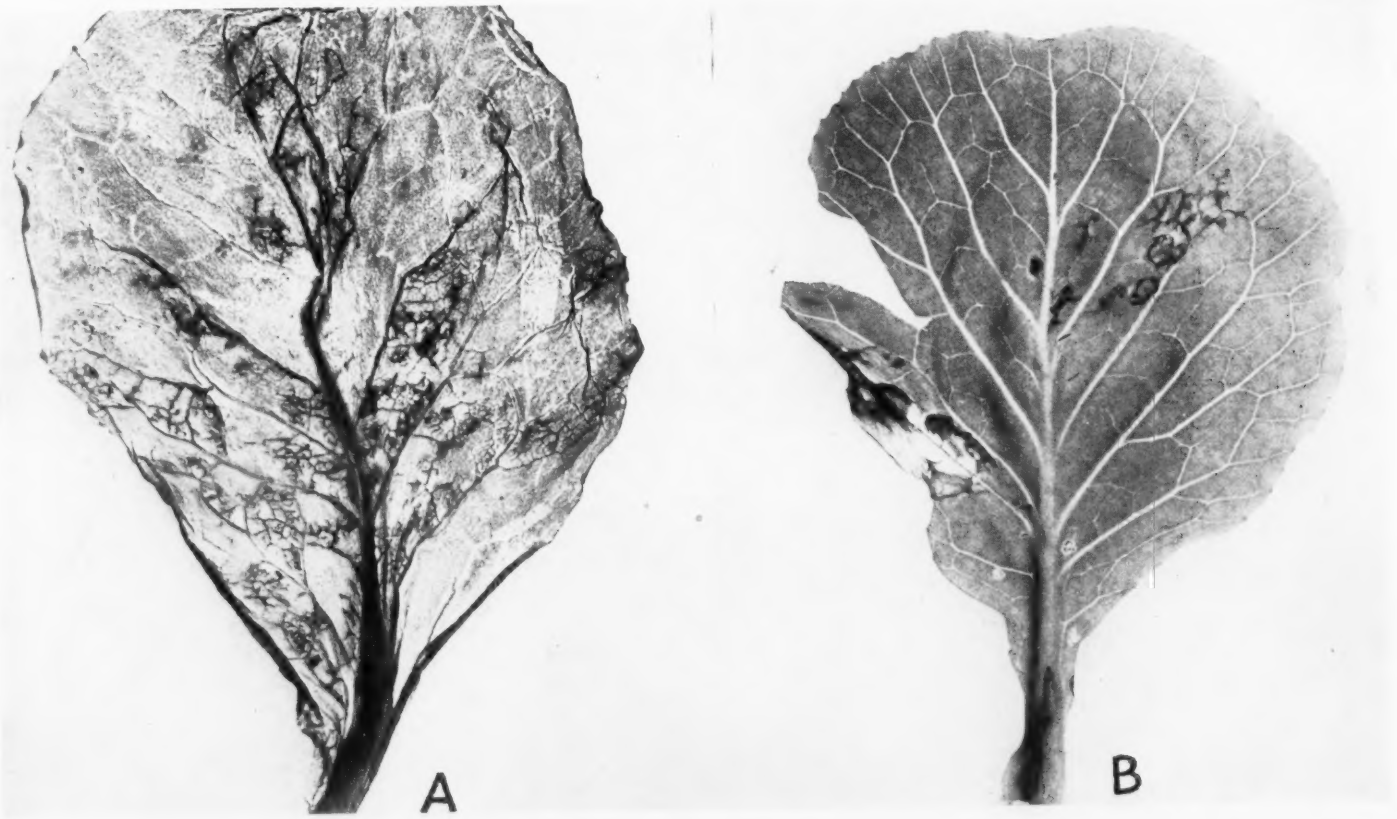


FIG. 3. *A*. Common cabbage leaf inoculated at base of midrib with *Bacterium campestre* isolated from Chinese cabbage. Photographed seventeen days after inoculating.

*B*. Common cabbage leaf inoculated at base of midrib with same cultures. Photographed two weeks after inoculating.

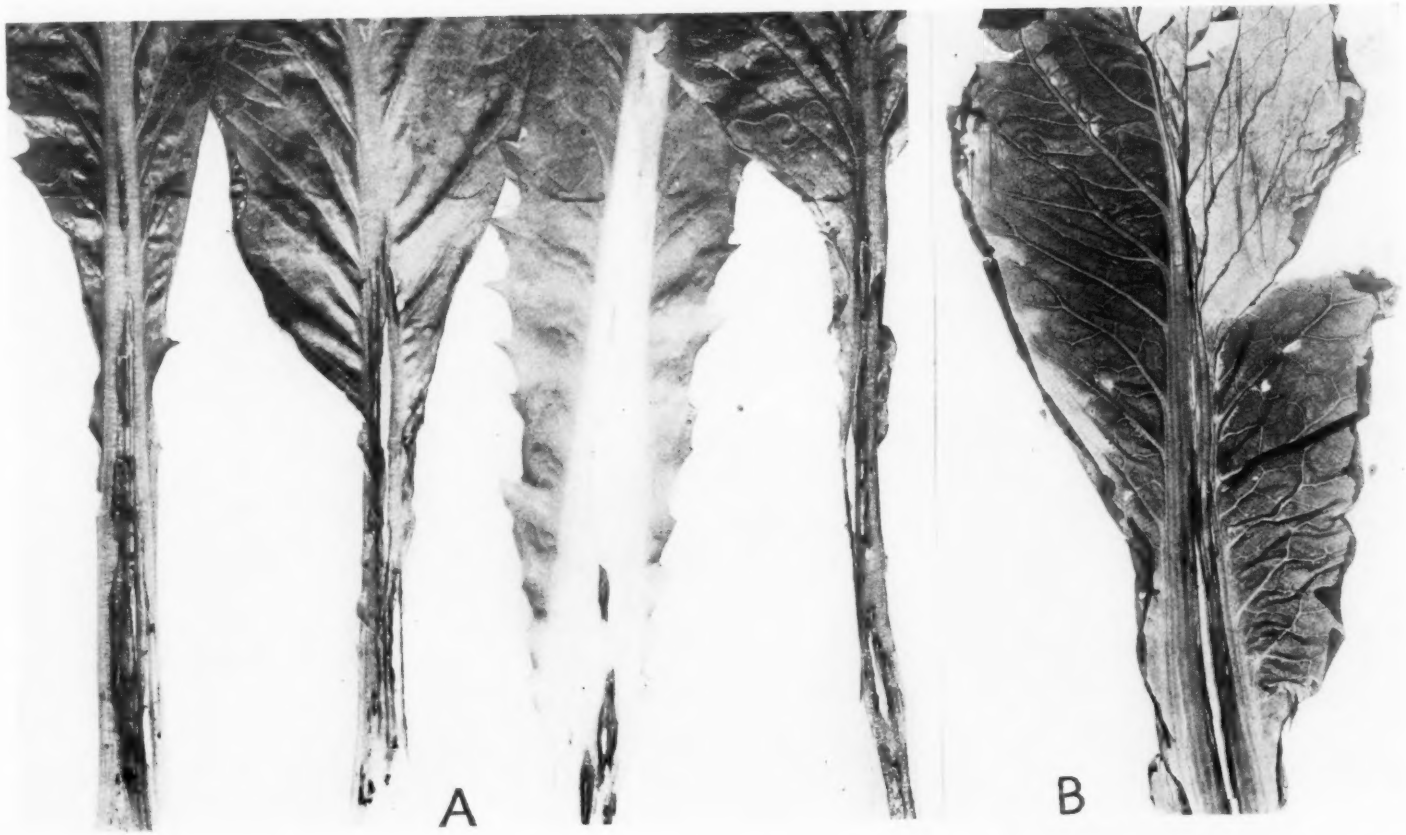


FIG. 4. *A*. Chinese cabbage leaves inoculated at base of midrib with *Bacterium campestre* isolated from Chinese cabbage. Photographed seventeen days after inoculating.

*B*. Chinese cabbage leaves inoculated at base of midrib with same isolations. Photographed nearly four weeks after inoculating.

INOCULATIONS

Inoculations were made into Chinese cabbage and common cabbage by spraying the plants with water solutions of the organism made from agar cultures; also by smearing the bacteria on the leaf surfaces and stems and puncturing with a sterile needle.

Infection was slow in the sprayed plants, two to three weeks being required. When the surfaces were smeared and punctured, infection took place in from four days to a week and continued to spread.

Infection has two lines of travel: from the water pores or other points of infection on the leaf down the blade to the stem; and when the stem is infected first through the fibrovascular bundles of the stem out into the midrib and blade. In the field, most of the infection seemed to be from the stem and roots or base of the leaf upward.

Fully 90 per cent of the inoculations took. The organism was reisolated from the browned midribs of the Chinese cabbage. There is some difference in the appearance of the infection of the two species of cabbage. In the Pai Ts'ai and Pak choi the brown veining does not show in the blade so definitely as it does in the common cabbage (figs. 3 and 4), nor is the discoloration so dark in the Chinese cabbages as in the common cabbage. However, the dark brown midrib in the Chinese cabbages, which may be an inch or more broad, is a very striking feature. Inoculations into the stem by punctures caused withering of the plant in two to four weeks, depending on the age and rate of growth of the plant.

The temperature of the greenhouse was around 78°F. day time and 65°F. at night, and the plants had plenty of moisture but not too much.

One set of puncture inoculations was made in a cooler house kept at 70°F. day time and 55°F. at night, and the amount of moisture reduced. At the end of a week there was no trace of infection in the plants at the lower temperature and reduced moisture, while those plants inoculated in the same way at the same time and kept in the warmer house with more moisture supplied showed good infection at the end of the week.

One set of inoculations was made by placing drops of a water suspension of the organism very carefully in the margins of the leaves of quickly growing plants of both Chinese and common cabbage. Darkening of the tiny veins was noted in the common cabbage in seven days but not until twelve days in the Chinese cabbage. The course of the disease continued to be more rapid in the common cabbage.

CULTURAL CHARACTERS

The causal organism isolated was at first thought to be a new one, as its appearance on the beef-agar plates did not seem to agree with the description of *Bact. campestre*. There are other cultural differences between it and *Bact. campestre* isolated from the common cabbage (4), (7) but these differences are not sufficient and the pathogenic features are enough alike to decide that we are dealing with *Bact. campestre* infectious to new species of Brassica, namely *Brassica pekinensis* (Lour.) Gagn. and *Brassica chinensis* Jusl.

The colonies on potato agar appear two to three days after pouring plates; they are at first cream colored; when a day old they are yellowish in reflected light, a little lighter colored in transmitted light, with fine markings on the surface. They are round, thickish, smooth shining, 2 to 4 mm., four days after pouring from diseased material. The youngest ones have no ring margin; the older ones a definite ring margin which shows radiate lines under the hand lens. The margin which is thinner and lighter colored than the rest of the colony is in some cases almost bluish. As the colonies get older they become a definite yellow color and some of them are a centimeter in diameter.

On beef agar the colonies are at first cream white, bluish in transmitted light, many with a ring margin. They become yellow as they grow older.

Sterile milk is cleared without coagulation, the casein being precipitated slowly, the clear whey becoming a light yellow color.

Litmus milk is blued slowly; in sixteen days it is nigrosin violet (Ridgway).

The organism has a distinctive growth on sterile potato cylinders. It is bright yellow at first, filling the water in the tube with a dense, yellow slime. Later the growth becomes a brown color. There is slow breaking down of the starch.

Beef gelatin is liquefied in two weeks.

No definite pellicle is formed in beef bouillon. There is no growth in Cohn's solution, no reduction of nitrates to nitrites, no production of gas or of indol.

The organism, unlike *Bact. campestre* isolated from the common cabbage, will grow in beef bouillon over chloroform. It is unlike it also in that it produces no indol while the common cabbage organism produces a slight amount; also in its negative action in Cohn's solution, for the common cabbage organism sometimes grows in Cohn's solution.

However, these cultural differences are no more significant than those differences often found in separate isolations of the same organism from the same species. Here we have isolations from two different species.

## TREATMENT

The treatment of the disease falls into the class of those diseases in which preventative measures have to be taken. The treatment of seed is recommended. It will have little value if the soil is infected, but less risk is run if the start is made with clean seed.

*Bact. campestre* is not a spore bearer but the vegetative rods seem able to survive long periods of drying.

Harding, Stuart, and Prucha in their work with common cabbage proved the presence of *Bact. campestre* on four lots of cabbage seed. Four black-rot infected seed-cabbage plants were threshed separately, the seed rinsed in sterile water, and cultures made. *Bact. campestre* was obtained in the cultures from all four lots and its pathogenicity proved by inoculations into cabbage plants.

They conclude that much of the seed on the market is contaminated with *Bact. campestre* and that some of these germs may survive the winter and infect the young cabbage plants that are produced in the spring. They recommend the soaking of seed fifteen minutes in a 1–1000 corrosive sublimate solution or in formalin 1 pound to 30 gallons of water.

It has been shown by their experiments that cabbage seed soaked in water to which a culture of *Bact. campestre* has been added, then the seed dried and put away in test tubes, had live bacteria present eleven months afterward (8). These bacteria produced the characteristic symptoms when inoculated into cabbage plants.

In the seed bed, soil should be used that is known to be free from disease. Otherwise, it should be steamed in advance or a fire built over it.

As moist conditions play an important part in the appearance and spread of this disease, a fundamental requirement in growing the Chinese cabbages is a well drained field.

Worms and insects are disseminators and they can be reduced in numbers by spraying with insecticides.

Infection from the soil has to be considered, for this can occur readily with rough handling of the seedlings at the time of transplanting. Should the organism be present in the soil, then the injured roots with exposed fibro-vascular bundles offer a ready entrance for the bacteria and root infection follows.

The removal of infected leaves from the plant even when the disease first makes its appearance in the field has not proved a successful method of combating the disease (2).

Care taken in the destruction of diseased plants will go a long way toward the prevention of further losses another year. Infected plants should never be left to decay but should be burned or thrown into a jar containing dilute, crude sulphuric acid, or mixed with fresh lime.

Thoughtless dumping of diseased plants on the compost heap to be used later for fertilizing may account for infection in new fields. It is not only gardeners, ignorant of the infectious nature of plant diseases, who do not practice sanitary methods with plants, but often those who are informed. Field sanitary methods cannot be emphasized too much.

## SUMMARY

The Chinese cabbages—both loose and tight-headed varieties—are subject to the black rot disease of the common cabbage produced by *Bact. campestre*, and like the common cabbage there may be also a rapid secondary infection by a soft rot organism.

The disease is very destructive in those seasons when there are high temperatures with excessive moisture.

The organism enters the plant through the water pores of the leaf and through the wounds in roots and stem. Insects also act as carriers.

The broad midribs of the Chinese cabbages turn a dark brown. Innumerable spottings may occur in the blade.

The soft rot organism destroys the heart at once. The spotting of the blade is unlike that due to the fungus *Alternaria brassicae*, which is a spotting confined to the loose outer leaves of the head.

Treatment is of a preventive character. Sanitary methods in the fields where the disease has occurred, spraying to destroy worms and insects that might act as carriers, careful handling of plants while transplanting them, and treatment of seed with corrosive sublimate 1–1000 for fifteen minutes or formalin 1 pound to 30 gallons of water are recommended.

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# A NEMATODE DISEASE OF RED CLOVER AND STRAWBERRY IN THE PACIFIC NORTHWEST

L. P. Byars

With Plates VI and VII

During the past summer the writer has observed a serious disease of red clover (*Trifolium pratense* Linn.) and strawberry (*Fragaria* sp.) plants in the Pacific Northwest, caused by the nematode *Tylenchus dipsaci* (Kühn) Bastian, (synonym *T. devastatrix*) which has long been known to attack many different species of plants in Europe but which has only recently been discovered to cause very severe damage in this country. Since the discovery of this nematode on hyacinths at the bulb propagating garden of the United States Department of Agriculture at Bellingham, Washington, in 1913, by the writer (1), Dr. N. A. Cobb[1] has observed it each successive year on specimens of red clover or strawberry plants sent in from other localities in the Northwest. During the past season Prof. Ralph. H. Smith (5) of the University of Idaho has found this parasite to be the cause of heavy losses to the red clover crops in many localities in Idaho, and it is also known to occur on this crop in eastern Washington and Oregon and in central Utah. In June of this year, the writer observed the nematode attacking strawberry plants growing on the experimental farm of the Oregon Agricultural Experiment Station, Corvallis, Oregon, where it had been seen three years previously by Prof. A. L. Lovett.[2] Since this article has been prepared the author has found the nematode on strawberry plants from Port Terrace, Oregon. These plants were collected in July, 1915, and sent to Dr. C. L. Shear, who preserved them in formalin and who kindly submitted them to the writer. Outside of the Pacific Northwest the nematode is known to have occurred in 1907 in Kansas, where Dr. E. A. Bessey (2) discovered it causing a stem disease of rye. Very probably it occurs in other parts of the United States and has been overlooked.

[1] Cobb, N. A., U. S. Department of Agriculture. Unpublished data.

[2] Lovett, A. L., Oregon Agricultural College. Letter to Dr. N. A. Cobb, dated July, 1916.

## DESCRIPTION OF THE DISEASE

*On red clover.* As seen in irrigated fields of Idaho and Utah, mature diseased plants are usually reduced in size and send up from the crown a number of adventitious shortened stems or shoots, which gives to badly affected plants somewhat of a witch's-broom or bush-like appearance, as shown in plate VI. The crowns of such plants generally show a dry rot, which extends for a short distance downward into the tap root and upward into the stems. Because of this internal rotting, the above-ground parts of these plants may be easily separated from the tap root by slight pulling. The stems are often swollen, particularly near their bases, may have their internodal spaces shortened, and are frequently bent near the end. Affected leaf stipules, petioles, and sometimes the leaves become distorted and show blister-like swellings. Badly diseased plants usually lose their normal green color, wilt, and die. However, there seems to be no characteristic discoloration of the foliage associated with the earlier stages of the disease.

*On strawberry.* Manifestations of the disease on strawberry may be seen on any part of the plant above ground. The plants as a whole are usually somewhat dwarfed, of a lighter green color than normal plants, and produce at the crown many adventitious branches. As shown in plate VII, affected leaves are distorted, wrinkled, swollen, and may be greatly reduced in size. On stems, petioles, stolons, fruiting branches, and at the base of leaves conspicuous galls which contain the nematodes in great numbers may be produced. This disease is different from the so-called "cauliflower" disease of strawberry described by Ritzema Bos (4) and caused by another nematode, an *Aphelenchus* sp., presumably *A. ormerodis* Ritzema Bos. Plants attacked by the latter nematode, which is much smaller than *Tylenchus dipsaci*, produce a large number of dwarfed adventitious branches, some of which are stimulated by the nematode to grow together throughout their length. Because of this stem thickening and fasciation, diseased plants resemble somewhat the upper portion of a cauliflower or pineapple plant. So far as known an *Aphelenchus* sp. has not been reported on strawberry in this country although one has been described on violets and other hosts.

## DISTRIBUTION AND DAMAGE

The disease on red clover has been found only in the Pacific Northwest and in this region only in the irrigated sections. Whether it is confined to clover that is grown under irrigation is an important question which should be answered as soon as possible. The writer has been unable

to find it on small plantings of clover in the neighborhood of Greeley, Colorado, Madison, Wisconsin, or Washington, D. C., but has not had opportunity to make even minor observations in other sections east of the Pacific Northwest.

Microscopic examinations by Dr. N. A. Cobb, Prof. Ralph H. Smith, and the writer show that the causal nematode in a living condition can be transported along with cleaned and uncleaned clover seed. It is highly probable that the parasite has been introduced into and spread within this country by this means. Spread in the irrigated regions no doubt has been largely by water.

General infestation of the clover in some of the irrigated sections of the Northwest has already taken place. This is particularly true in the Twin Falls, Idaho, district, where it is difficult to find an uninfested field and where the damage has been so severe that many farmers the past season have plowed up their clover and planted other crops.

Although preserved specimens of the disease on strawberry, collected at Port Terrace, Oregon, have been seen, the trouble on this plant has been found in the field at only one point, Corvallis, Oregon. At the latter place none of the plants were dying from the trouble at the time of examination although much damage in the way of reduced yield and lessened quality of fruit was apparent. On strawberry the trouble is very probably most commonly spread by means of the young infected plants which are used for propagating purposes.

## CAUSE

The disease of both the red clover and strawberry is caused by the nematode *Tylenchus dipsaci* (Kühn) Bastian, which usually attacks only the above-ground parts of plants. The eggs, larvae, and adults of this organism in great numbers may be seen with the aid of a hand lens if infected tissues are teased apart in a few drops of clear water. The adults are spindle-shaped, milky-white eelworms of little more than 1 mm. in length. In a desiccated condition the parasite, in its earlier stages of development, may remain motionless but alive for more than a year.

Often closely associated with the nematode on clover are certain other organisms, which seem to assist in only a secondary manner in causing the trouble. These are a small mite and the larvae of two insects, which were found constantly about the tap root and crown of affected plants and which have been previously thought to be the primary cause of the trouble. The writer has, however, just completed inoculation experiments in the greenhouse and laboratory which prove that the nematode alone is responsible for inducing the disease on clover. Details of the

results of these experiments, which are being continued, will be published later.

Because of a lack of opportunity, inoculation experiments designed to demonstrate that the nematode is the sole cause of the disease on strawberry have not been carried out, but on affected plants in the field no other parasite could be found.

## HOST RELATIONS

No consistent morphological differences between the nematode on strawberry and red clover could be found by the writer. That there may be physiological differences, however, seems probable since rows of strawberry plants free from infection were observed growing in fields of clover which were severely diseased. Likewise disease-free crops of beans, alfalfa, and rye, which have been reported as hosts in Europe, were seen in fields that had grown a severely infected crop of clover the previous season. These observations harmonize with the view held by Ritzema Bos (3), namely, that in Europe this nematode becomes specialized on certain of its hosts and will not readily attack any other. The possible development of biological strains of the organism is an important and interesting question which must be solved before practicable methods of control can be well devised or carried out. Until this has been accomplished it would seem inadvisable to plant on infested land any of the crops which are known to be seriously damaged by the nematode in Europe. Some of the more important of these are: beans, potatoes, onions, peas, alfalfa, and most of the clovers, vetches, and grains. The writer has found the nematode attacking only one wild plant, a *Physalis* sp., which was growing in a badly diseased field of clover in Utah. Plants of this genus apparently have not been previously reported as a host (5).

Active investigations of the disease are under way in the laboratory and greenhouse, and field studies in coöperation with the Idaho Experiment Station have been planned.

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## PLATE VI

A Nematode-Infected Red Clover Plant from Provo, Utah

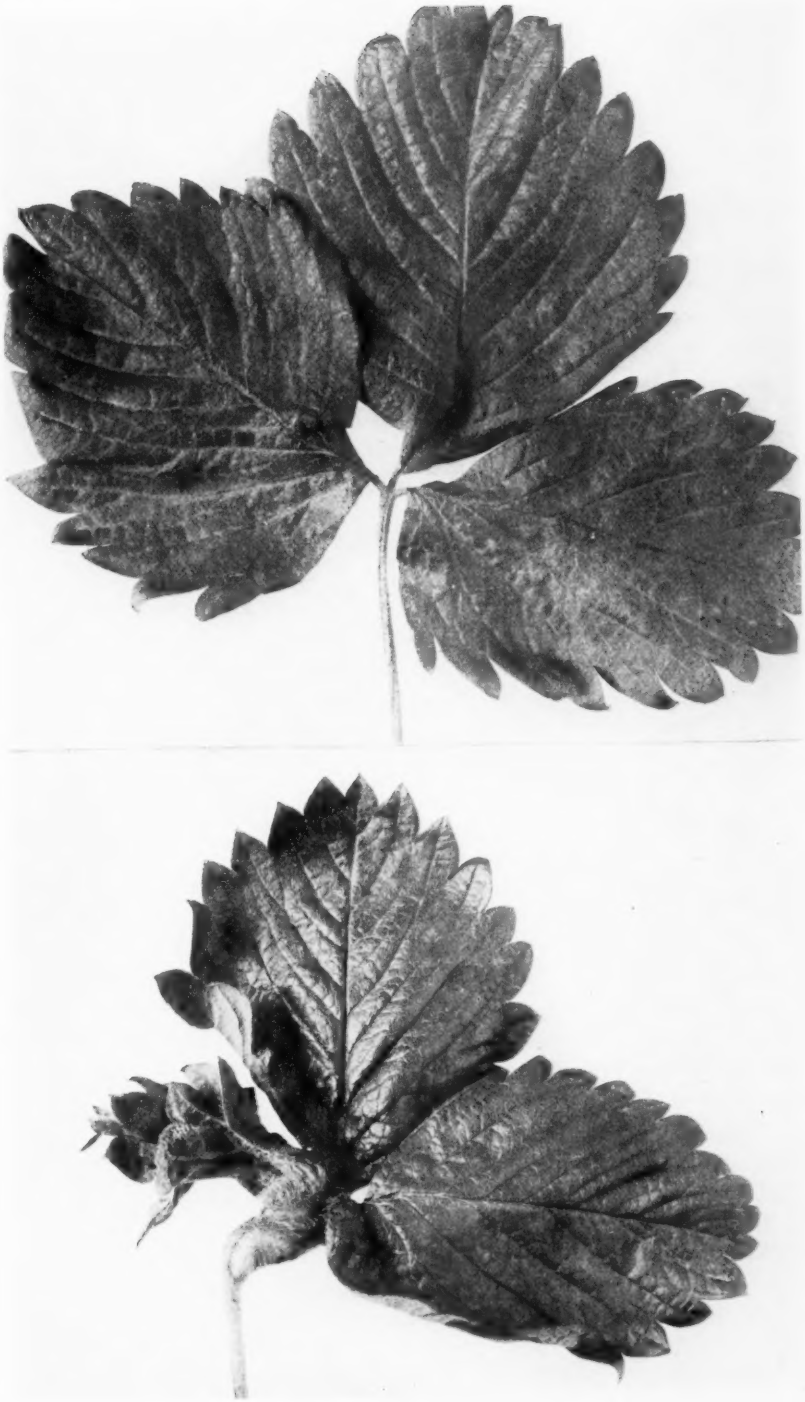
Photographed natural size. Note the adventitious, dwarfed branches, some of which have swollen terminals.

BYARS: NEMATODE DISEASE OF CLOVER AND STRAWBERRY

## PLATE VII

A Normal (Above) and a Nematode-Infected (Below) Leaf of Strawberry from Corvallis, Oregon

Photographed natural size. Note on the diseased leaf the distorted, dwarfed leaflet and the nematode gall on the petiole.



BYARS: NEMATODE DISEASE OF CLOVER AND STRAWBERRY

# STREAK DISEASE OF POTATO

W. A. Orton

With Plate VIII and One Figure in the Text

In the summer of 1912 the writer studied a disease of potato that occurred in Maine, New York, and Wisconsin, and prepared a description which was intended to be published under the name "potato streak" in a bulletin on "Potato Wilt, Leaf-Roll, and Related Diseases."[1] This article was withheld because the nature of the streak disease had not been demonstrated and it was hoped that further laboratory work would result in the discovery of a parasitic causal organism. Our efforts in this direction were unsuccessful, and it now seems desirable to record our early observations, defining the characteristics of this interesting potato malady, which is being found from time to time in various parts of the country. There are somewhat similar obscure troubles which it is desirable to differentiate from the streak for clearness in discussion, even though it is possible that future research will reveal a relationship.

Potato streak is a disease of the foliage, attacking full grown and otherwise normal and healthy plants and frequently progressing rapidly to destroy them.

## CHARACTERISTICS

So far as our observations go, streak is first to be detected on the upper, full grown leaves of the potato plant, in the form of elongated or angular spots following the veinlets and invading the parenchyma (fig. 1). While somewhat more conspicuous on the upper side, these spots are also to be seen on the under side as narrow, discolored streaks along the veins. The typical form may very quickly be recognized and distinguished from the spots caused by early blight (*Macrosporium solani*), though the more diffuse spots of the streak resemble slightly the Macrosporium spots, particularly when the latter also occur on the same plants, as is often the case.

The destructive progress of the streak is rapid. Shortly after its appearance on the leaflets, as described, the petiole will be found affected and slightly discolored by longitudinal streaks. The petiole collapses and the leaf then withers and hangs limp, or the petiole breaks at the point of attachment to the stem and hangs by a thread in a dead and

[1] Orton, W. A. Potato Wilt, Leaf-Roll, and Related Diseases. U. S. Dept. Agr. Bul. 64, 48 p., 16 pl. 1914.

dried condition. Since it is the full grown leaves that are first attacked, there is at this stage a circle of dead leaves about a third of the way down from the terminal shoot, while the latter is still green (plate VIII, fig. 1).

FIG. 1. POTATO STREAK

Drawing from nature of the early stage of streak from experimental plots in northern Maine. J. F. Brewer.

The hanging dead leaves are quite characteristic of the disease, while all the characters mentioned form a picture that is quickly seen and well remembered.

A prominent characteristic of streak is the brittleness of the affected parts. The leaves break off very easily and the stem is also brittle. This symptom is, however, also met with in other potato diseases, such as curly-dwarf and mosaic dwarf.

The stem begins to turn brown and dies at a point below the tip. The upper leaves then wilt and die, and the disease progresses downward. Long, faint brown streaks appear on the stems also. Apparently the discoloration is just below the epidermis but not in the vascular bundles. These brown stripes are not continuous from leaflet to petiole, nor from petiole to stem. There is no discoloration of the vascular bundles in the lower part of the stem or root, nor are there any other evidences of disease at the root. The several stalks in a hill die separately; all stages may be observed in one hill, from the first spotting of the leaves to quite dead stalks. A late stage is illustrated in plate VIII, figure 2.

No effect on the tubers has been found. The yield is reduced in proportion to the time of onset of the disease. The first cases were found when the Maine field was visited, August 9, 1912. At this time several plots were dead and the disease was quite generally distributed. The New York field was visited August 16 and 17, at which time the disease was under full headway. A considerable portion of the field was dead, and as all stages of streak could be traced, it was concluded that this new disease was the principal cause of the early death of the foliage, particularly in the seedling collection of the Bureau of Plant Industry which was being grown in both states. In other parts of the field there was a good deal of tipburn and early blight.

## VARIETAL RESISTANCE

Marked differences in varietal susceptibility exist. This was most prominent in the collection of seedling varieties where streak was most prevalent. Some plats remained entirely healthy, while others directly alongside were destroyed. More streak has been observed in European varieties, such as Factor, than in American sorts. The seedlings referred to were for the most part hybrids with European varieties of potatoes, and this may account for their greater susceptibility. Outside of this seedling collection, the most prominent case of streak the writer has observed was a field of several acres of Factor at Puyallup, Washington, in September, 1914. It was observed that year at Jerome, Idaho, but this also in seedling varieties. A very typical case was observed in a field of Rural New Yorker near Madison, Wisconsin, in 1912.

The nature of the streak disease remains in doubt. Its appearance and general course present striking similarities to some of our well known bacterial diseases, such as *Ps. malvacearum* on cotton and *Bact. michigensis* on tomato.

The writer was confident that bacteria would prove to be the cause, but efforts to isolate an organism have thus far been futile. For the most part plates poured from the discolored areas remained sterile. Colleagues have made similar attempts, with similar negative results. It is believed, nevertheless, that eventually an organism will be found and shown to cause potato streak.

### OTHER CONFUSING LEAF-SPOTS

It will be noted that the illustrations presented show streak on plants of normal size and development, and this is typical of the disease as the writer knows it.

It should not be confused with the spotting and streaking of potato leaves associated with advanced stages of leaf-roll or mosaic dwarfs, or from weak or otherwise degenerate plants. Such spots and streaks are common on moribund potato plants, but the pathological picture is quite different from that described above.

There is, for example, a disease not uncommon in our trial grounds, marked by weak, erect stems, from which frequently the spotted leaves fall prematurely, from the base upward, so that a palm-tree effect is produced.

It is quite within the possibilities that streak may attack a plant suffering from mosaic or other disease, and that when we know the cause of streak we shall be able to differentiate it more clearly from other leaf spots. The point to be emphasized now is that streak is not per se the result of some other disease, but is quite distinct and separable from all other potato diseases known to the writer.

To one observing the rapid spread of this disease through plats of susceptible varieties and its destructive effect, it would appear potentially dangerous to the potato industry, but seven years have passed since it was first observed, and the disease is still something of a rarity. This may be taken to indicate that our standard varieties of potatoes possess substantial resistance to streak and that it is not to be greatly feared by commercial growers.

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### PLATE VIII

FIG. 1. Potato plant, unnamed seedling variety, dying from streak. Note dead leaves hanging in center where streak developed first.

FIG. 2. Potato streak, advanced stage. This plant was normal and healthy two weeks earlier.

FIG. 1

FIG. 2

# INFECTION EXPERIMENTS ON TOMATOES WITH PHYTOPHTHORA TERRESTRIA SHERB. AND A HOT WATER TREATMENT OF THE FRUIT

J. ROSENBAUM

Phytophthora terrestria Sherb. on tomatoes has been under observation in southern Florida for the past three years. During the early season of 1918–1919 it was hard to find a single fruit infected by this fungus in one of the tomato fields, while observations made in various parts of this same field later, following some exceptionally heavy rains, showed an infection of more than 66 per cent of the fruit, by actual count.

Sherbakoff,[1] referring to the rot caused by this fungus, states: "The rot occurs almost invariably only when the fruit touches the ground or is very close to it." In view of the high percentage of infection found in the field, it was supposed that if tomatoes rested or were placed on this infested soil, a high percentage of infection would result. A few experiments were performed, the results of which were somewhat contrary to what might have been expected. They give some additional information on infection, and suggest a possible treatment of the fruit for this disease in its very early stages.

## EXPERIMENT I

The purpose of the experiment was to determine whether contact with diseased soil is sufficient to produce a high percentage of infection.

Two hundred healthy, large green tomatoes were picked from a field known to have little if any Phytophthora, kept in the laboratory for two days so as to allow any infections present to develop, and then buried in an infested portion of the field in such a way that some of the tomatoes were only partially covered while others were two inches below the surface. There was no doubt that the land where the tomatoes were placed was infested, as counts made there a week earlier showed about 90 per cent to be infected.

The tomatoes were removed in small lots at twenty-four hour intervals, examined for any signs of infection, wrapped in paper and kept at room temperature, and again examined later. After a period of two weeks 14 per cent of the 200 fruits had become infected.

[1] Sherbakoff, C. D. Buckeye Rot of Tomato Fruit. Phytopathology **7:** 119–129. 1917.

The percentage of infection was much lower than was expected in view of the heavy natural infection at first found. The soil during the time of the experiment was relatively moist, but no rains occurred.

### EXPERIMENT II

The purpose of this experiment was to determine the influence of water on infection of the tomato fruit when in contact with diseased soil.

Flower pots were filled with infested soil obtained from the row where Experiment I was made, and one or more healthy, green tomatoes were placed in each pot and lightly covered with the soil. The pots were divided into three lots of twelve each. The treatments and the results obtained are summarized in the following table.

| LOT NUMBER | TREATMENT | PERCENTAGE OF INFECTION |
|---|---|---|
| 1 | Immersed in water for sixteen hours | 84 |
| 2 | Watered each day | 23 |
| 3 | Not watered | 0 |

The experiment was repeated in the laboratory and the results obtained were similar to the above. To determine whether the amount of water in the soil will influence infection in the field in a similar manner, two lots of ninety-six tomatoes each were buried in an infested field as outlined in Experiment I, in adjacent rows but separated by a soil ridge. One lot was watered each day. At the end of eight days both lots were removed. The lot watered had developed 63 per cent infection, while the adjacent lot had developed only 10 per cent.

These experiments leave little doubt that an abundance of water is necessary for an epidemic infection in the field. This is to be explained by the fact that moist infested soil containing mycelium of the fungus is not as infectious as infested soil where the zoospores are liberated and can swim about freely. Laboratory inoculations likewise have repeatedly shown that the zoospores can cause infection much more readily than any other stage of the fungus. The zoospore can penetrate and produce an infection on healthy, green fruit without previous injury.

### EXPERIMENT III

Working on the theory that the zoospores are liberated in the soil when an abundance of water is present, it was of interest to determine the effect of slight traces of copper in the water, when applied to infested soil.

One or more healthy tomatoes each were placed in infested soil in pots as for Experiment II. These were divided into several lots. The treatment and results are shown in the following table:

| LOT NUMBER | TREATMENT | PERCENTAGE OF INFECTION |
|---|---|---|
| 1 | Not treated | 3 |
| 2 | Watered heavily | 34 |
| 3 | Immersed in water for thirty-six hours | 55 |
| 4 | Immersed for thirty-six hours in water containing 1–5000 $CuSO_4$ | 0 |

Stronger solutions of copper sulphate were used but no infection resulted in any case. A weaker solution than 1–5000 was not tried.

Immediately following the heavy rains referred to in the first paragraph, a number of growers picked their tomatoes. It was found that, notwithstanding the fact that most of these did not show any signs of infection at the time of picking, they developed the buckeye rot, due to *Phytophthora terrestria*, after being packed. In one case a grower was compelled to repack 500 crates on account of the rot, and less than one-third were found healthy.

One of the largest growers in southern Florida has made it a practice during the last three or four years to wash tomatoes in hot water before shipment. The method has given results that have led to the installation of such washing tanks in several packing houses along the East Coast. It was on this account that the following experiment with hot water treatment was made.

### EXPERIMENT IV

The purpose of the experiment was to determine the efficiency of hot water in preventing the spread of new and subcuticular infections of Phytophthora on tomato fruit.

In order to obtain fruit in the earliest stages of infection, a lot of green, healthy tomatoes were brought to the laboratory, washed, immersed in a 1–1000 mercuric chloride solution for five minutes, and placed in large moist chambers. They were inoculated in these chambers with a decoction of swarm spores, by means of an atomizer. Part of the inoculated fruit was removed approximately at the end of each twenty-four-hour period. Each lot of fruit as it was removed was divided into two parts, one of which was immediately immersed in hot water, while the other was left untreated. From previous preliminary experiments it had been found that green tomatoes can withstand water at a temperature of

60°C. for one and one-half minutes. If treated at 65°C. for the same length of time an occasional fruit will develop a rusty color and show slight shriveling, indicating injury from the hot water. Therefore, all the infected fruit was treated for one and one-half minutes at 60°C.

The following table shows the results obtained.

| NUMBER OF HOURS BETWEEN ORIGINAL INOCULATION AND REMOVAL OF FRUIT FROM CHAMBER | PERCENTAGE OF TREATED FRUIT DEVELOPING ROT | PERCENTAGE OF UNTREATED FRUIT DEVELOPING ROT | REMARKS |
|---|---|---|---|
| 24 | 0 | 100 | No infection was evident when fruit was removed |
| 46 | 0 | 100 | The infection was evident at time of treatment |
| 71 | 0 | 100 | As above |
| 96 | 66 | 100 | As above |
| 122 | 100 | 100 | As above |

The results of the experiment indicate that: (1) a period of *not more* (and possibly less) than twenty-four hours is necessary for the swarm spores to be in contact with the tomato fruit to produce infection; (2) immersing infected fruit in hot water at 60°C. for one and one-half minutes will prevent decay of infected fruit which does not yet show macroscopic signs of the lesion or those in which the tissues have not yet been penetrated deeply. In the experiment performed 71 hours after the inoculation was made the lesion was macroscopically evident; nevertheless, the hot water treatment stopped its further progress. Ninety-six hours after inoculation was made it was not possible to kill the fungus in the tissues in every case. One hundred and twenty-two hours after inoculation the invading mycelium had penetrated too deeply into the tissues to be affected by the hot water treatment.

The good results said to be obtained by some of the growers from treating fruit with hot water may be ascribed to the fact that not only are fungus spores and sand removed from the surface, but in addition it may also kill new infections which have not penetrated very deeply into the tissues of the fruit.

Repetition of all the experiments outlined here gave similar results.

SUMMARY

An abundant moisture supply, preferably free water, in diseased soil, is a necessary condition for producing an epidemic infection with *Phytophthora terrestria* Sherb. on tomatoes. This is to be explained by the fact that only under such conditions will an abundance of zoospores be liberated.

The addition of 1–5000 copper sulphate solutions to infested soil will prevent infection of green tomatoes when in contact with such soil.

The immersion of green tomatoes in water at 60°C. for one and one-half minutes will prevent the spread of the buckeye rot where the infection is very recent and the fungus has not penetrated deeply into the tissues.

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## BRIEFER ARTICLES

### PHONY PEACHES: A DISEASE OCCURRING IN MIDDLE GEORGIA[1]

DAVID C. NEAL

WITH PLATE IX AND ONE FIGURE IN THE TEXT

Phony peaches, apparently a new disease of the peach, has been known to exist for some time, especially in the peach belt of middle Georgia. In fact, the distribution of the disease appears to be more or less limited to that peach section of the state. This rather peculiar trouble of peach trees, although observed a few years ago by growers, as well as by Federal and State pathologists, has received practically no investigation, with the result that the factors concerned in its cause remain quite obscure.

The disease has been given the tentative name of "phony peaches" by growers, probably because of the more or less dwarfed character of trees which are affected. It seems appropriate to state that orchardists and pathologists have heretofore confused the phony disease and another somewhat similar trouble of the peach, namely, "collar-edema." The latter has been studied by Mr. M. B. Waite of the Office of Fruit Disease Investigations, Bureau of Plant Industry, and according to him the collar-edema disease of peaches is entirely distinct from the phony disease occurring in this state. Waite's[2] description of the collar-edema disease of peaches is as follows: "It is characterized by an edema or abnormal swelling of the collar of the tree, mostly below the ground line. The bark is thicker and fleshier or has a larger proportion of the flesh part than normal. The lenticels are enlarged and the wood is more or less brittle or corky, or less fibrous than normal. The trees are sometimes snapped off by the wind because of their brittle character. The growth is short and stubby and the leaves become yellow and drooped and usually fall prematurely." According to the same author, the collar-edema disease is restricted mainly to the eastern section of the United States, extending into New York State. It has also been observed in the State of Michigan. The phony disease of peach, however, has thus far been seen by the writer only in middle Georgia. With the exception of the short, stubby growth of the twigs, none of the above symptoms are evident on trees having the phony disease.

[1] Published with the approval of the Georgia State Board of Entomology.

[2] From correspondence supplied the Georgia State Board of Entomology.

## DESCRIPTION OF THE DISEASE

### *Leaf symptoms*

A tree affected by the phony disease is readily detected, on account of its leaf symptoms. The leaves are somewhat larger than normal, and are especially marked by their dark green color. In typical cases this vivid green color of the foliage is so pronounced that such a tree is easily distinguished from normal ones by an observer passing an orchard, even though several paces away (plate IX, figs. 1 and 2).

### *Twig symptoms*

The twigs of trees having the phony disease are particularly noticeable because of their short, stubby growth. In cases where the current season's growth of normal shoots would average $2\frac{1}{2}$ to 3 feet or more, a tree affected by the phony disease seldom averages one-third or one-half of this growth (fig. 1). As a result, the entire head of the tree is reduced to a rather compact shape, owing to the forshortened internodes and resulting close spacing of the leaves. With such malformations induced, the trees usually fall rapidly behind normal ones on the same soil as to growth and size.

### *Cause of the disease*

The present experimental work with this disease is so limited that the factors which are responsible for the symptoms which have been briefly described above are as yet unknown. The phony disease is possibly one of the so-called physiological diseases, falling perhaps in the same category with troubles like peach rosette, peach yellows, and the mosaic diseases of many plants. Since the disease is known to occur over a wide range of soil types, its relationship to such factors as soil fertility, soil moisture, malnutrition, or excessive physiological stimulation as the result of certain plant foods must all be studied under laboratory and field conditions before definite causes can be set forth.

### *Effect of the disease on fruit development*

Personal observations and conversations with growers who are familiar with the disease indicate that the amount of fruit which a phony tree sets is very small. Frequently fruit on such trees attains almost normal size, but the period of ripening is usually considerably retarded. There is some variation among the different varieties in this respect, but the fruit matures at least ten or twelve days later than normal, is insipid in taste, and has little, if any, market value.

*The relation of age of trees to the development of the disease*

From field observations it appears that trees which have reached bearing age are more susceptible. Many cases are encountered among those which have attained four and five years, but the largest areas affected are found in orchards which are much older (five to ten years). The youngest case which we have on record is that of a tree which developed typical symptoms of the disease during its second season's growth.

FIG. 1. COMPARISON OF THE CURRENT SEASON'S GROWTH OF NORMAL AND DISEASED SHOOTS

Healthy shoots at left, diseased shoot at right. Photographed August 16, 1919.

*Varietal susceptibility*

The existence of varietal susceptibility or immunity does not seem to be established. In fact, most of the varieties of the peach which are grown in middle Georgia have been found to be affected to a certain degree. The disease is prevalent on Elberta, Hiley Belle, Belle of Georgia, Mayflower,

and J. H. Hale. It no doubt attacks other varieties. Owing to the frequent occurrence of the disease in plantings of Elberta, one is sometimes led to consider this variety more susceptible. On the other hand, it constitutes the largest number of trees of any single variety planted in this section, and this fact alone may account for the large number of such trees affected by the disease.

*Presence of the disease on seedlings*

The phony disease has been found in the vicinity of Fort Valley, Georgia, on seedlings which are now in their sixth season's growth. Owing to this discovery, the nursery phase of the problem immediately presents itself for careful investigation. The following points appear to be extremely important in this connection, and are now being actively investigated by the writer. First, does the disease have its origin to any extent in the nursery, especially in the localities where nursery stocks are propagated for this section? Second, is the phony disease, like yellows and peach rosette, transmittable by budding?

Pending the outcome of experiments which are now in progress bearing upon this particular phase of the problem, it would appear to be only the part of sound judgment for the orchardist and the nurseryman to familiarize themselves with this relatively little known peach disease. They should certainly exercise the greatest caution in their selection of budwood from those regions which are known to be infested, in order to avoid disseminating the trouble to other localities which are free of the disease.

GEORGIA STATE BOARD OF ENTOMOLOGY
THOMASVILLE, GEORGIA

## PLATE IX

FIG. 1. A peach tree of the J. H. Hale variety now in its sixth year, showing typical symptoms of the phony disease. Note the short, stubby growth of branches and the dark green, compact foliage. Photographed August 16, 1919.

FIG. 2. A healthy tree of the same variety and of the same age growing in an adjoining row. Photographed August 16, 1919.

FIG. 1

FIG. 2

## PHYTOPATHOLOGICAL NOTES

*Late seasonal production of aecia of Cronartium ribicola.* A freshly matured aecium of *Cronartium ribicola* was observed by the writer on a native tree of *Pinus strobus* near Amery, Wisconsin, September 16, 1919. It had developed at the upper end of a lesion, which had resulted from a natural infection and which had borne eight normal sized aecia early in the season. The lesion was on a branch which was about ½ inch in diameter at the place of infection.

The aecium was of recent origin since the newly ruptured peridium was filled with bright orange-colored spores. Dr. Perley Spaulding, of the Bureau of Plant Industry, Dr. S. B. Fracker and Mr. H. J. Ninman, of the Wisconsin State Entomologist's Office and Prof. John W. Stephens, of the University of Syracuse were with the writer at the time the aecium was found. Mr. Ninman stated at this time that one or two days previously he had seen two similar aecia in the same locality. The writer observed a newly developed aecium of *Cronartium ribicola* near Littleton, N. H., July 21, 1918. Aeciospores of this fungus are known to be carried many miles by the wind. Puzzling occurrences of this fungus on Ribes late in the season, which had been earlier apparently free from disease, may be explained by the presence of such late aecia. Judging from the observations of Miss Dosdall (Phytopathology **8**: 619, 1918) it seems that spores of such late seasonal development as mentioned above may over-winter in the aecia and become a source of early infection in the following season.

H. H. York.

*Personals.* Mr. H. F. Bain has been appointed pathological inspector for the Federal Horticultural Board, U. S. Department of Agriculture, to take charge of the treating of seeds and plants in connection with the inspection work at Washington, D. C. Mr. Bain is a graduate of Brown University and has recently received his discharge from the Army.

Dr. John P. Benson has resigned his position as assistant pathologist in the Office of Cotton, Truck, and Forage Crop Disease Investigations, Bureau of Plant Industry, to become assistant professor of agricultural extension in the University of California. He is now detailed as farm adviser to Fresno County, with headquarters at Fresno.

Mr. Chas. R. Hursh and Mr. A. A. Potter, formerly assistant pathologists in the Office of Cereal Investigations, Bureau of Plant Industry, have left the Departmental service, the former to take up graduate study at the University of Chicago and the latter to take up work in the agricultural division of the Bureau of the Census.

# LITERATURE ON PLANT DISEASES[1]

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COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

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